less frequently preceded by convulsions. Probably the chief reason of the less frequent occurrence of convulsions from this cause in the human subject is, that the amount of air accidentally admitted is less, and death consequently is less rapid than when air is forcibly blown into the vein of an animal. It would probably be found, on a careful inquiry, that the occurrence of convulsions in these cases depends upon the circulation being suddenly and completely arrested.

It has been noted, in some cases of suddenly fatal pulmonary embolism, that death has been preceded by convulsions; and Virchow observed, amongst the results of artificial embolism of the pulmonary artery in animals, convulsions and dilatation of the pupil. (*Des Emboles Pulmonaires, par* B. Ball, page 129.)

We find, then, a large amount of evidence pointing to the conclusion that sudden and extreme anæmia of the brain will cause epileptiform convulsions, and a theory of epilepsy has been framed in accordance with these facts; the theory being that the cerebral anæmia, which is the immediate cause of the convulsion, is the result of spasm of the cerebral arterioles. It may be said with truth that this is only one step towards an explanation of the phenomena, and that the cause of the arterial spasm remains to be determined. We will presently revert to this question.

It is, I think, pretty generally admitted that this theory of cerebral anæmia from arterial spasm is quite consistent with the phenomena of epilepsy. It is a matter of general observation that, at the very commencement of an epileptic fit, the face is pallid. There is obviously anæmia of the superficial vessels, and with this there is probably associated anæmia of the intracranial vessels which supply the brain itself. The pallor is in most cases soon succeeded by lividity, owing to the venous engorgement which results from impeded respiration and pulmonary circulation. It is very remarkable that, while the face is pallid, the heart is beating strongly and the carotids throbbing violently. These phenomena would be explained by extreme contraction of the muscular arterioles, resisting the escape of blood from the arterial trunks into the capillaries.

Kussmaul and Tenner endeavoured to support the theory of arterial spasm by experiment, and to some extent they succeeded. In each of two white rabbits they ligatured the two subclavians and one carotid; the cervical sympathetic, on the other side, was then exposed and galvanised, with a view to excite contraction of the arterioles by the stimulus conveyed through the vaso-motor nerves. In two animals no effect was produced; but in the third the background of the eye became completely pale; the pupil dilated, so that the iris could scarcely be seen; the neck was drawn back, and violent convulsions occurred. The electrodes being removed, the spasms ceased, the pupil contracted, and the background of the eye became red; but the animal continued in a swooning condition. After some minutes, electricity applied to the sympathetic nerve produced the same effect as at first. A third attempt to excite convulsion did not succeed.

The authors suggest that these experiments deserve repetition, with a view of rendering certain what at present is probable, namely, "that epileptic convulsions can be brought about by contraction of the blood-vessels induced by the vaso-motor nerves."

According to this theory, then, epilepsy is the result of sudden anæmia of the brain; and this anæmia, when not caused by a sudden and profuse hæmorrhage, or by some impediment to the circulation outside the cranium, is due to an extreme contraction of the muscular arterioles. This arterial contraction may be determined by two main classes of causes:

1. By a purely nervous reflex influence, such as, for example, may be excited by anger or terror, by the irritation of the gums during dentition, by a calculus in the kidney, the ureter, or the gall duct, or by worms in the intestines.

2. In the second class of cases, a blood-poison is the exciting cause of the arterial spasm and the resulting epileptic convulsion. This includes all cases in which convulsions result from retained excreta, of which uræmic convulsions are a typical example.

From the preceding narrative of facts, it appears to be highly probable that uræmic convulsions are directly due to a sudden and extreme anæmia of the brain, resulting from contraction of the cerebral arterioles, and that the arterial contraction is excited by the influence of impure blood upon the vaso-motor nerves and centre.

This theory, moreover, indicates two modes in which uræmic convulsions may be prevented, namely: first, by means directed towards removing the morbid quality of the blood; and, second, by remedies which lessen the reflex excitability of the nervous centre.

(*To be continued.*)